Effect of vibration on antagonist muscle coactivation during progressive fatigue in humans

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1. Biceps femoris antagonist coactivation increases during progressive fatigue. Our purpose was to determine if the mechanism that increases coactivation during fatigue is susceptible to vibration. Vibration drives α-motoneurons via the Ia loop, producing force without descending motor drive, and thus uncoupling antagonist and agonist activation. Evidence that vibration increases coactivation disproportionately from its 'common drive' would suggest the possibility that some of the effects of fatigue are mediated through a segmental reflex loop.

2. Ten male subjects performed repeated maximal voluntary isometric contractions (MVCs) of the knee extensors of one leg. Paired submaximal test contractions (50% of MVC), without visual feedback, were performed when MVC reached 85, 70 and then 50% of its initial value. Vibration was applied to the patellar tendon during one test contraction in each pair.

3. Vibration reduced test contraction force below control values. However, coactivation increased at the same rate in both conditions. Biceps femoris coactivation was greater during vibration, but did not change during fatigue in either condition.

4. Our observations suggest that agonist–antagonist muscle pairs are controlled as a single motor unit pool by a common central drive. Vibrating the agonist increases antagonist coactivity, but does not alter the rate at which coactivation increases during fatigue. This supports the idea that agonist coactivation is controlled by a central mechanism.

In the early part of the century, Sherrington (1906) described the process of reciprocal inhibition that occurs when Ia afferents are stimulated and excite homonymous motoneurons while simultaneously inhibiting the antagonist via Ia inhibitory interneurons (Tyler & Hutton, 1986). According to this doctrine, voluntary contractions are accompanied by complete antagonist inhibition (Tyler & Hutton, 1986). Sherrington also discussed the phenomenon of double reciprocal inhibition in which antagonist muscle pairs are simultaneously active (Tyler & Hutton, 1986). This is now referred to as coactivation and in most voluntary contractions it produces force that acts in opposition to the agonist (Patton & Mortensen, 1970; Solomanow, Baratta, Zhou & D'Ambrosia, 1988; Carolan & Cafarelli, 1992; Psek & Cafarelli, 1993). The central nervous system (CNS) shifts between reciprocal inhibition and coactivation of antagonist muscle, depending on the circumstances (Brooks, 1986).

Recent studies of coactivation in our laboratory arose from the search for non-hypertrophic neuromuscular adaptations that contribute to the increase in the maximal force-generating capacity of resistance-trained muscles. We have found that resistance training of the knee extensors results in less antagonist coactivation (Carolan & Cafarelli, 1992). This reduction in opposing force contributes to the small but significant increase in extension maximal voluntary contraction (MVC) after 1 week of training (Carolan & Cafarelli, 1992). Similarly, during progressive fatigue of the knee extensors, antagonist coactivation increases and contributes to the loss of extensor force-producing capacity (Psek & Cafarelli, 1993).

The idea that coactivation is governed by a central descending common drive was first presented by DeLuca & Mambrito (1987). They hypothesized that the CNS controls agonist and antagonist muscle as a single motoneuron pool, causing both to be simultaneously active. Recently, Nielsen & Kagamihara (1992) suggested that there is a specific motor programme for coactivation and that interneurons in the reciprocal pathway are actively inhibited from a central source during coactivation. This depression of the reciprocal

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inhibition leads to a high excitability level in the motoneurons of the antagonist muscles and results in coactivation. The depression of the reciprocal inhibition is centrally mediated, but its cause is unknown. Nielsen & Kagamihara (1992) suggest several possibilities: (i) presynaptic inhibition of Ia afferents increases during coactivation and depresses the inhibition; (ii) descending facilitation of Renshaw cells depresses Ia inhibitory interneurons; and (iii) Ia interneurons receive inhibitory inputs from both segmental and suprasegmental sources (Hultborn, 1976). Increased activity of any one of these sources could explain the depression.

The results of our previous experiments (Carolan & Cafarelli, 1992; Psek & Cafarelli, 1993) are compatible with the hypothesis that coactive muscles are driven from a common descending drive (DeLuca & Mambrito, 1987; Nielsen & Kagamihara, 1992). Vibration excites the spindle primaries which, in turn, drive the α-motoneurons of the homonymous muscle, thereby partially uncoupling the common drive to the agonist–antagonist muscle pair (Eklund & Hagbarth, 1966). The purpose of the present investigation was to determine if vibration produced any evidence that a peripheral pathway also plays a role in the increase in coactivation during progressive fatigue.

METHODS

Experimental model and subjects

Ten male subjects (age 23·5 ± 0·6 years, height 176·8 ± 2·3 cm, weight 74·6 ± 3·7 kg; data are means ± S.E.M.) participated in the experiment. They reported low-to-average levels of physical activity and were not currently involved in any physical training. The procedures were approved by the York University Human Participants Review Committee. All subjects gave written informed consent.

Protocol

Two days prior to data collection, subjects practised maximal extension and flexion contractions until MVC measurements were repeatable within 10% of each other. Before beginning the protocol, a tonic vibration reflex (TVR) was produced in the resting limb of both legs in order to test the effectiveness of vibration in producing Ia input to the motoneuron pool. The initial data were obtained while the subjects performed at least three brief (3–5 s) flexion and extension MVCs, each separated by 2 min (Fig. 1). The largest MVC was used to normalize all force data. Superimposed shocks (8 pulses at 50 Hz) were delivered to the muscle during two of the MVCs to verify maximality. Maximal integrated electromyography ($IEMG_{max}$) values were averaged from the recordings accompanying the three highest force records within 10% of each other. Test contractions were each held for 15 s with 2 min in between.

The protocol was always applied to the dominant limb and divided into four stages of fatigue. The initial data were obtained prior to the fatiguing contractions and stages 1–3 were defined as the points at which MVC had reached 85, 70 and 50% of its initial value. Fatigue was induced by a series of repeated extension MVCs, held for 3 s with a 50% duty cycle. When MVC had fallen to 85% of its initial value (stage 1), two test contractions at 50% of MVC were performed. Vibration was applied during one of these contractions. This sequence was repeated when MVC had fallen to 70% (stage 2) and 50% (stage 3) of the initial value. The alternating cycles of contraction and relaxation were maintained by following a pattern of target torque production and rest drawn on an oscilloscope screen.

To avoid fatigue in the leg not engaged in the protocol, surface electrodes were placed over its vastus lateralis during the

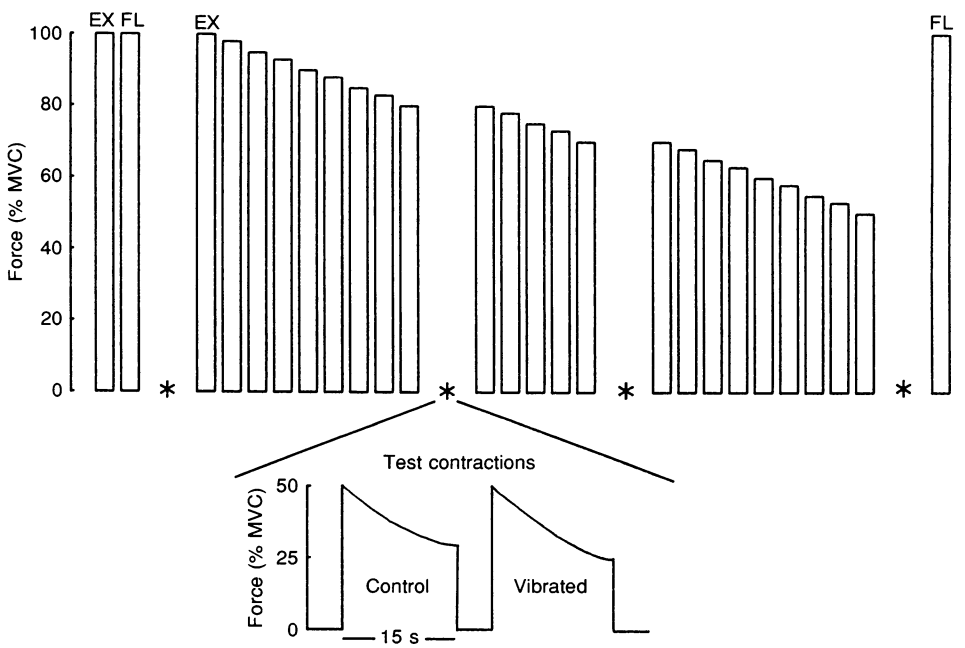

Figure 1. Schematic representation of the experimental protocol
EX, extension MVC; FL, flexion MVC.

experimental session, and the EMG was displayed on an oscilloscope. Activation of the unfatigued leg during experimental leg contractions could be detected and discouraged.

Vibration

The vibrator, a modified engraving tool (model 332-01; 1500 Hz; 1·5 mm amplitude; Dremel Moto-Flex, Racine, WI, USA), was secured over the patellar tendon with non-extensible straps. Vibration excites the Ia spindle endings and, to a lesser degree, tendon organs and Pacinian corpuscles (Burke, Hagbarth, Lofstedt & Wallin, 1976; Cafarelli & Kostka, 1981). Moreover, it drives the α-motoneurons without an accompanying increase in central drive (Matthews, 1964; Vallbo, 1979). Vibration results in a gradual contraction beginning 1–3 s after the onset of the stimulus and a gradual decline after it is removed (Sherrington, 1906). In these experiments, vibration was necessary in order to provide additional Ia input without augmenting central command (Bongiovanni & Hagbarth, 1990). The neuromuscular system is thus perturbed at the segmental level and any changes in coactivation as a result of vibration would be via peripheral feedback mechanisms.

Test contractions

The test contractions were held at 50% of MVC for 15 s without visual feedback about force production. This was accomplished by covering the oscilloscope screen once the subject had reached the target force. Isometric contractions held at a constant level of force sensation with no visual feedback results in an exponential decline in force as a function of time that is highly repeatable within and between subjects (Cain & Stevens, 1973; Pandolf & Cain, 1974). Vibration of the patellar tendon during these contractions results in a more rapid decline in force than control. It is well known that subjects can voluntarily suppress the TVR when they can see the force record (Eklund & Hagbarth, 1966). Therefore, contractions performed without visual feedback were necessary in order to avoid suppression of the TVR.

Force

Subjects were seated in a leg dynamometer which had been modified to allow access to the hamstrings (Psek & Cafarelli, 1993). The chair was tilted backward 45 deg with the subjects' knees flexed at 90 deg and the hips restrained with a seat belt. The legs were secured to strain gauges which registered the forces applied during leg extension or flexion. This signal was amplified (×10), and viewed on a 20 MHz digital storage oscilloscope (BK Precision, Dyanscan Corp.). Stimulating pads were placed over the proximal and distal portions of the quadriceps and the hamstring muscle groups of both legs. A train of eight 75 μs supramaximal pulses were delivered at 50 Hz to the agonist muscle during MVCs at the end of each stage to verify that the muscle was maximally activated (Merton, 1954). If it was not, the protocol was continued. This method is based upon the technique described by Merton (1954) and modified by Bigland-Ritchie, Furbush & Woods (1986).

Electromyography

Bipolar, silver–silver chloride surface electrodes (E.Q. Inc.) were placed over the vastus lateralis 12–15 cm from the superior patellar border and over the belly of the long head of biceps femoris. Potentials were preamplified (×40) at the electrode and then passed through a second stage amplifier (frequency response flat from 1–1 kHz). All signals were stored on FM tape during the experiment and digitized downstream through an A/D board (DT2801) at 1024 Hz into a computer (486 DX/33 MHz). The EMG signals were full-wave rectified and integrated (IEMG) using sequences in EasyestLx software (Keithly Asyst Technologies, Taunton, MA, USA). Maximal IEMG ($IEMG_{max}$) values were obtained by integrating a 1 s epoch of the rectified EMG signal that preceded maximal force output by 40 ms. Test contraction IEMG values were obtained from the last 8 s of the 15 s contraction.

Statistical analysis

The MVC data from extension and flexion contractions were analysed with two-factor, repeated-measures analyses of variance, with time (initial and stage 3) and limb (fatigued and unfatigued) as the independent variables. This was done to determine differences in MVC force, IEMG and antagonist IEMG between limbs and over time.

Data from the test contractions of the fatigued leg were compared to determine differences between the force, agonist and antagonist IEMG between conditions and over time. This was done with a 2 × 4 repeated-measures ANOVA with condition (vibrated and control) and test period (initial and stages 1, 2 and 3).

Data from the test contractions of the unfatigued limb were analysed with a 2 × 2 repeated-measures ANOVA with condition (vibrated and control) and test period (initial and stage 3) as the independent variables. Probability levels were set at 0·05 for all analyses, pooled data are expressed as means ± S.E.M., and significant differences were tested with Tukey *post hoc* tests.

RESULTS

Tonic vibration reflex (TVR)

The amplitude of the TVR and its associated EMG were repeatable and not significantly different within subjects from day to day and between the orientation and the experimental sessions. There were no significant differences in force and vastus lateralis IEMG during vibration between the fatigued and unfatigued legs. Force measured during TVR was 3·0 ± 0·28% of MVC but vastus lateralis IEMG was 10·8 ± 0·52% of maximal IEMG ($IEMG_{max}$). The discrepancy between force and IEMG occurred because the apparatus was tilted backward at 45 deg for the subject's comfort. Thus the weight of the leg had to be overcome before force could register on the strain gauge. When the apparatus was set at 90 deg, the force production matched IEMG activity at 10–12% of MVC, which is consistent with previous work from our laboratory (Cafarelli & Kostka, 1981). Despite this discrepancy, none of the absolute forces reported in this paper have been adjusted for the position of the dynamometer because they would have no effect on the relationships of interest.

Fatigue protocol

Figure 2 shows the averaged maximal extension force, vastus lateralis $IEMG_{max}$ and biceps femoris $IEMG_{max}$ obtained during the fatiguing protocol. There was a main effect of time on extension force ($P < 0·001$) and vastus lateralis $IEMG_{max}$ ($P < 0·003$). Extension MVC declined by 47·4% by the end of the protocol. On average, it took 12·6 ± 3·8 contractions to reach stage 1, another 16·8 ± 3·6 contractions to reach stage 2, and 28·5 ± 7·1 contractions

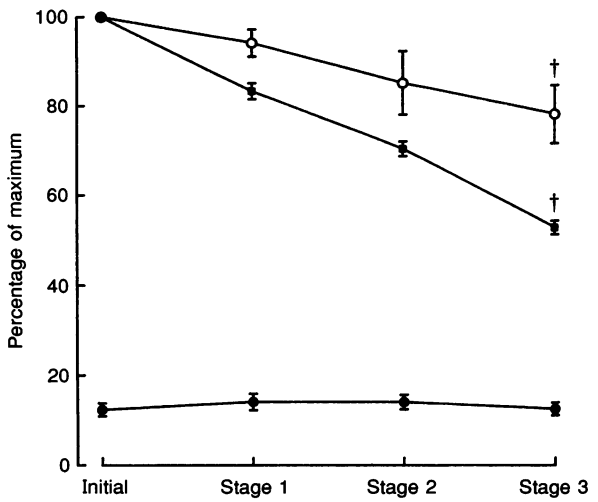

Figure 2. Some effects of the fatiguing protocol
Extension MVC (■) and vastus lateralis $IEMG_{max}$ (○) declined over the course of fatigue. Biceps femoris IEMG (coactivation; ●) did not change. † indicates main effect of time; $P < 0{\cdot}0001$.

to reach stage 3. Vastus lateralis $IEMG_{max}$ declined by 22% by the end of the protocol. Despite the fatigue in the extensors of the fatigued leg, flexion MVC and biceps femoris $IEMG_{max}$ of the fatigued leg were not different at the beginning and end of the protocol (Fig. 3). Extension MVC, vastus lateralis $IEMG_{max}$, flexion MVC and biceps femoris $IEMG_{max}$ of the unfatigued leg were also unchanged from their initial values (Fig. 3).

Test contractions

Fatigued leg

No significant interactions were observed in any of these analyses. There were significant main effects of time ($P < 0{\cdot}001$) and condition ($P < 0{\cdot}001$) on extension force and a main effect of time ($P < 0{\cdot}001$) for vastus lateralis IEMG (Fig. 4, top panel). Force in the vibrated condition was about 16% less ($P < 0{\cdot}05$) than control levels at the

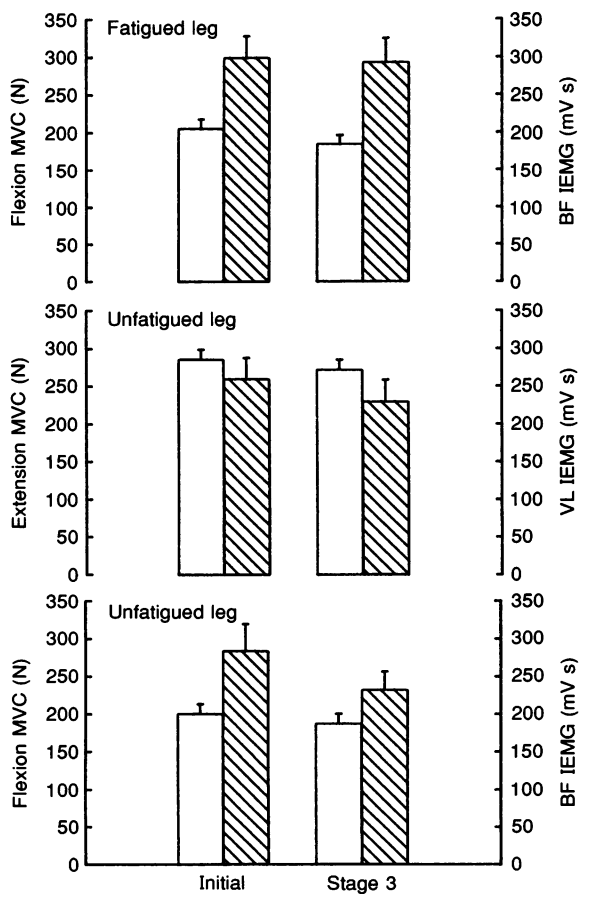

Figure 3. Effects of fatiguing protocol on flexion MVC and biceps femoris $IEMG_{max}$
Despite the fatigue evident in the extensor muscles of the fatigued leg, flexion MVC (□) and biceps femoris (BF) $IEMG_{max}$ (▨) were not different before and after the fatigue protocol. Extension MVC and vastus lateralis (VL) $IEMG_{max}$ in the unfatigued leg were also not different before and after the fatiguing protocol. Similar results were obtained for flexion MVC and biceps femoris $IEMG_{max}$ of the same leg, which indicates an absence of fatigue in this limb.

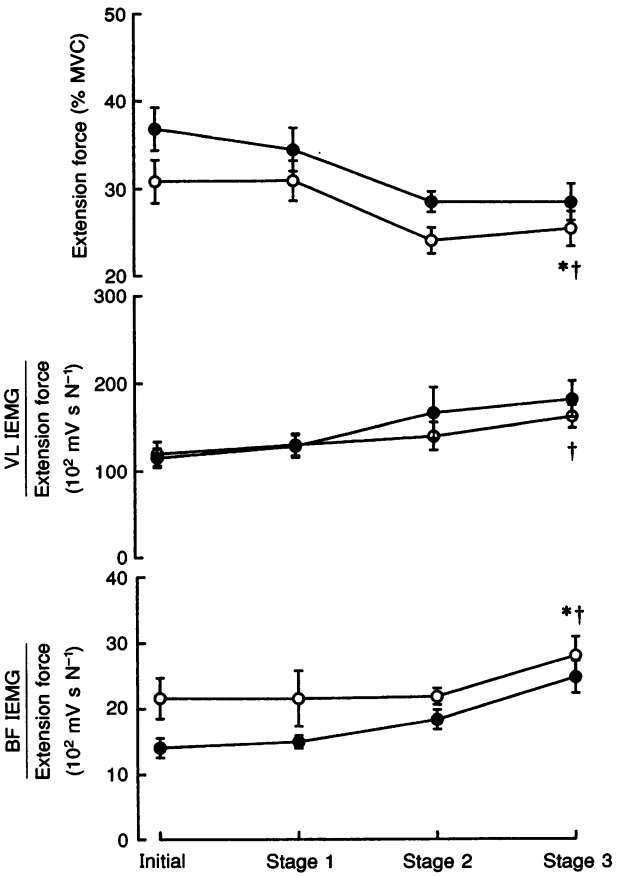

Figure 4. Response of the fatigued limb to the test contractions

Mean extension force declined over time in both conditions and was less in the vibrated condition than control at each stage of fatigue. There was no difference between conditions in the degree of vastus lateralis activation (VL IEMG) even though VL IEMG increased as a function of time. There was a greater amount of biceps femoris coactivation (BF IEMG) in the vibrated condition and coactivation increased in both conditions over the course of fatigue. * indicates main effect of condition, † indicates main effect of time; $P < 0{\cdot}05$. ○, vibrated condition; ●, control condition.

initial stage. The test contractions declined in both conditions and vastus lateralis IEMG per unit of extension force increased significantly as fatigue progressed (Fig. 4, middle panel). There were main effects of time ($P < 0{\cdot}002$) and condition ($P < 0{\cdot}012$) on biceps femoris IEMG. By the final stage of exercise, biceps femoris IEMG per unit of extension force had increased by 36% in the control condition and 10% in the vibrated condition ($P < 0{\cdot}05$, bottom panel). Vastus lateralis IEMG and biceps femoris IEMG are expressed per unit of extension force in order to normalize the data to extension force, which was different between conditions. When coactivation is expressed as the ratio of biceps femoris IEMG to vastus lateralis IEMG (BF IEMG/VL IEMG), there is a main effect between conditions but not over time on BF IEMG/VL IEMG ($P < 0{\cdot}004$) (Fig. 5). This illustrates that there was more coactivation when the patellar tendon was vibrated, but that vibration had no effect on the increase in coactivation during the fatiguing protocol.

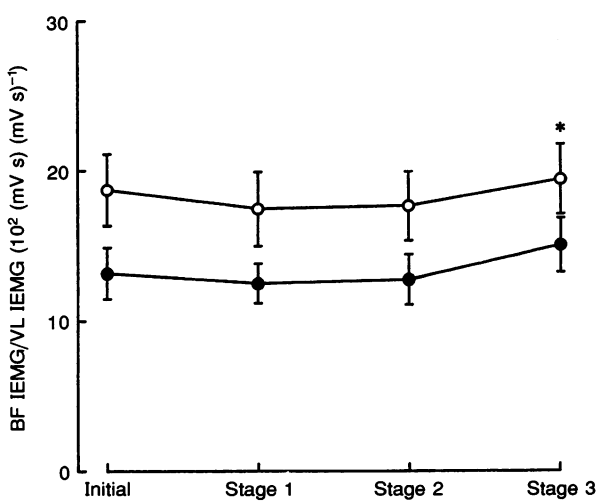

Figure 5. Effect of fatiguing protocol on coactivation ratio

The coactivation ratio (BF IEMG/VL IEMG) was greater during vibration due to a greater amount of biceps femoris IEMG. The coactivation ratio did not change during progressive fatigue in either condition. * indicates main effect of condition; $P < 0{\cdot}05$. ○, vibrated condition; ●, control.

Unfatigued leg

Figure 6 shows a main effect of condition on extension force during test contractions of the unfatigued leg ($P < 0{\cdot}001$, top panel). There was 21% less force produced during vibration than in the control condition at both the initial stage and at stage 3. There was a main effect of time ($P < 0{\cdot}007$, second panel from top) for vastus lateralis IEMG, which was greater at stage 3 than at the initial stage. There was a main effect of condition ($P < 0{\cdot}005$, second panel from bottom) in biceps femoris IEMG per unit of extension force. Thus there was more coactivation during vibration than during control conditions before and after the protocol. There was a main effect of condition ($P < 0{\cdot}007$, bottom panel) on the coactivation ratio, BF IEMG/VL IEMG, which was larger during vibration than during control conditions before and after fatigue (Fig. 6).

Sham experiment

There was some concern that vibration might be transmitted to the hamstring tendons through the straps that held the vibrator in place. Precautions such as additional padding around the knee were taken to avoid any transmission. We also performed a sham experiment, similar to the one described by Cafarelli & Kostka (1981). In this experiment the vibration was applied to the medial side of the knee joint and the vibratory effects on the IEMG of the quadriceps and the hamstrings were observed under different submaximal extension forces. There was no effect of vibration on the hamstring IEMG.

DISCUSSION

The purpose of this investigation was to seek evidence of a peripheral pathway capable of mediating the increase in coactivation during progressive fatigue. The results showed that biceps femoris coactivation increased during fatigue and that there was more coactivation during vibration of the patellar tendon than there was during the control condition. However, the coactivation ratio (the ratio of BF IEMG/VL IEMG during knee extension) did not change during the course of fatigue in either condition. Although vibration perturbs the coactivation ratio, we observed no evidence that the *increase* in coactivation during fatigue is mediated through a peripheral pathway.

Possible mechanisms responsible for the effects of vibration on coactivation

Vibration is thought to inhibit the contraction of antagonist muscles via Ia inhibitory interneurons (Eklund & Hagbarth, 1966). Our data, together with those of Jones & Hunter (1985), are not consistent with this idea since

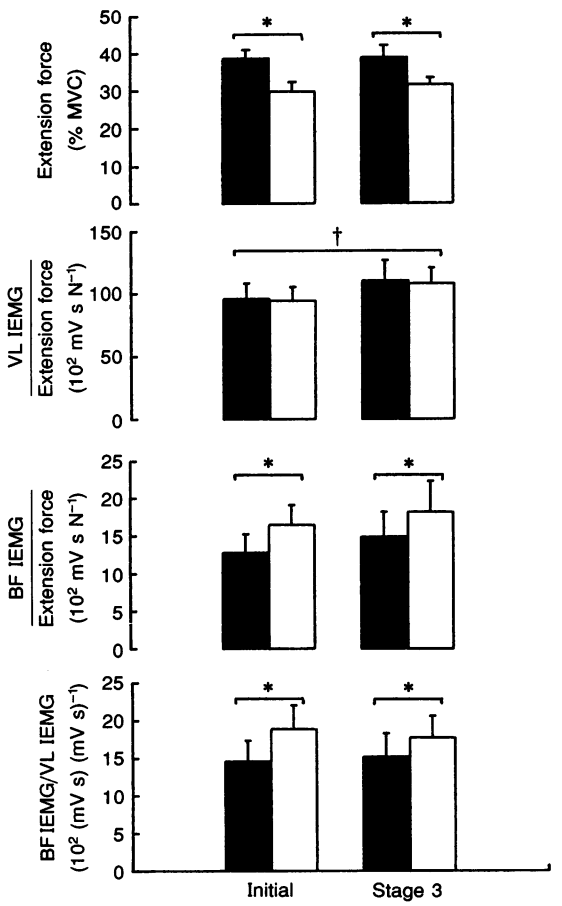

Figure 6. Response of the unfatigued limb to the test contractions

Mean extension force was less in the vibrated condition than control before and after the fatiguing protocol. There was no difference between conditions in the degree of vastus lateralis activation (VL IEMG), even though VL IEMG had increased after the protocol. There was a greater amount of biceps femoris coactivation (BF IEMG) in the vibrated condition, which resulted in a higher coactivation ratio (BF IEMG/VL IEMG) in the vibrated condition than in the control. * Indicates main effect of condition, † indicates main effect of time; $P < 0{\cdot}05$. ■, control; □, vibrated.

vibration resulted in a greater amount of antagonist coactivation. There are at least four explanations of how this change in coactivation could occur. (i) Since the TVR has a delayed onset, but the response of spindles to vibration is abrupt, the TVR may be difficult to explain purely on the basis of spindle mechanisms (Burke *et al.* 1976). (ii) Golgi tendon organs are more responsive to vibration during isometric contractions, which means they are more likely to activate antagonist motoneurons (Burke *et al.* 1976; Vallbo, 1979). (iii) The subjects had difficulty controlling force production during vibration. This could have increased coactivation since it is needed to control precise movements (Smith, 1981). Therefore, proprioceptive feedback could play a role in increasing coactivation. (iv) Driving α-motoneurons with vibration may increase excitation of Renshaw cells, which would inhibit Ia inhibitory interneurons, and increase coactivation (Brooks, 1986).

Coactivation during progressive fatigue

In our laboratory we have found that there is a constant ratio between the degree of activation of the extensors and flexors during isometric extensions of the knee (Carolan & Cafarelli, 1992; Psek & Cafarelli, 1993). Moreover, the ratio is maintained during progressive fatigue even though agonist drive increases steadily (Psek & Cafarelli, 1993). The constant coactivation ratio during fatigue in the present experiment shows that although vibration uncoupled the relationship between the two muscles, it had no effect on the relationship over time. Furthermore, comparing the responses to the test contractions of the unfatigued limb to those of the fatigued limb showed that vibration increases the coactivation ratio but is not associated with an increase in this ratio over time. Since coactivation increased as a result of fatigue but the coactivation ratio remained unchanged over the course of time, one may argue that the increase in coactivation as a result of progressive fatigue is centrally mediated.

If agonist and antagonist muscles are controlled as a single motoneuron pool, there must be a point in the pathway that reduces antagonist activation to about 10–20% of its original level, thus producing coactivation. If both muscles received an equal amount of drive, there would be no net movement of the limb. A reduction in antagonist activation must occur before the motoneuron pool in order for the agonist muscle to cause movement. It may be that this 'reduction point' is part of the central program for coactivation. Alternatively, it could also be segmental inhibition that is gradually released during fatigue, which would then cause an increase in coactivation. Nielsen & Kagamihara (1993) argue that according to their H-reflex data, it is a central phenomenon.

During coactivation, interneurons are uncoupled from parallel control of their corresponding motoneurons by a specific central command, thereby allowing the activity of the antagonist to increase in parallel (Nielsen & Kagamihara, 1992). This uncoupling would ensure a high excitability of both motoneuron pools and hence the development of force in both muscles (Nielsen & Kagamihara, 1992). The actual mechanism for coactivation has several possible sources, which hinge on interneuronal inhibition. The interneurons could be directly inhibited from a central source and, therefore, the increase in coactivation as a result of fatigue would be part of the central coactivation programme. Facilitation of Renshaw cell inhibition of the Ia interneuron would indirectly depress reciprocal inhibition and favour coactivation (Nielsen & Kagamihara, 1992). Ia inhibitory interneurons can receive input from several sources, including centrally mediated commands from the cerebellum and spinal neurons such as Ia afferents, making reciprocal inhibition and coactivation variable (Angel, 1977; Brooks, 1986). The most likely candidate appears to be an increase in presynaptic inhibition of Ia terminals on antagonist motoneurons (Nielsen & Kagamihara, 1992). Presynaptic inhibition originates centrally and increases during coactivation. This increase may serve as a damping mechanism, allowing excitability of the antagonist motoneuron to increase in parallel to the agonist motoneuron, which would serve to reduce the risk of oscillations in the agonist–antagonist muscle control system. Nielsen & Kagamihara (1993) also suggest that oscillations in this motor control system in an unstable situation, in which coactivation is required to ensure stability, may endanger the balance of the subject.

Conclusions

Resistance training reduces coactivation during submaximal isometric contractions and reduces coactivation in the untrained ipsilateral leg of the same person (Carolan & Cafarelli, 1992). This suggests that coactivation is controlled with a central programme. It is also known that there is an increase in coactivation during fatiguing isometric contractions (Psek & Cafarelli, 1993). This increase in opposing force detracts from net force production and contributes to fatigue. Psek & Cafarelli (1993) showed that vastus lateralis and biceps femoris EMG are correlated during progressive fatigue of the knee extensors, which suggests that coactivation is controlled by a central mechanism. The present experiment supports the findings from that study. Our data show that coactivation increases during fatigue and that vibration can uncouple agonist and antagonist motor drive. However, the failure of vibration to alter the rate at which coactivation increases during fatigue suggests that this process is centrally mediated.

Coactivation increases during fatigue by what appears to be a central mechanism that increases presynaptic inhibition of the Ia afferents conveying the inhibition to antagonist motoneurons. More coactivation may be a preventative strategy against intense unilateral forces that could damage

the knee joint. Coactivation decelerates the limb to avoid excessive strain on the joint capsule by powerful agonist activation. In so doing, force production is compromised since coactivation contributes to fatigue (Psek & Cafarelli, 1993). Coactivation also safeguards the agonist muscle against complete exhaustion by reducing the time to the limit of endurance. Therefore, the agonist muscle is not absolutely exhausted and may still perform at low levels of activity until fully recovered. Finally, avoiding oscillations in the agonist–antagonist motor control system would ensure stability and stiffness in conditions that require coactivation.

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